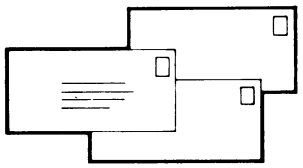

Letters to the Editor

Letters to the Editor are welcomed and will be published, if found suitable, as space permits. Submission of a Letter to the Editor constitutes permission for its publication in the Journal. Letters should not duplicate similar material being submitted or published elsewhere. Letters referring to a recent Journal article should be received within three months of the article's publication. The editors reserve the right to edit and abridge letters, to publish replies, and to solicit responses from authors and others.

Letters should be submitted in duplicate, double-spaced (including references), and should not exceed 400 words.

Elimination of Cotinine from Body Fluids

The brief article by Jarvis, *et al,*[1] in this issue of the Journal deserves comment. Their study was designed to examine the elimination of cotinine from body fluids and to compare the results with those of others. Their use of ex-cigarette and current cigar smokers complicates the methodology, and the use of nicotine capsules raises problems which make it difficult to compare this study with those which have utilized cigarette smoke.

Cigarette smoke contains a large number of chemicals which are strong inducers of hepatic enzymes and can alter the metabolism of nicotine and other exogenous compounds.[2-4] Further, the rate of nicotine uptake is dependent on the route of administration.[5-7] The nicotine administration selected by Jarvis, *et al,* fails to consider the lung as a site of xenobiotic metabolism.[8]

Although the authors state that limited data are available on the half-life of cotinine, two studies[9,10] have been reported by Kyerematen, *et al,* one of which (included in the author's discussion)[9] shows significantly longer plasma t ½ of both nicotine and cotinine in nonsmokers when compared to smokers after intravenous administration of ^{14}C-nicotine.[3] The second study demonstrates a significantly longer t ½ for nicotine in naive versus habituated smokers.[10] The latter study also compares pipe smokers, snuff dippers, and cigarette smokers, and suggests that the mode of uptake influences the rate of nicotine elimination. At least four other studies have demonstrated an extended t ½ for cotinine in passive smokers.[11-13]*

Finally, the authors state that the "nonsmokers in our sample had slightly shorter half-lives than the smokers . . ." Who are the smokers? According to their methods section, there are no cigarette smokers in the study, only ex-smokers, two of whom are current cigar smokers. Russell states that "cigarette smokers who switch to become secondary pipe or cigar smokers continue to inhale," and that secondary pipe and cigar smokers cannot, therefore, be regarded as ex-smokers.[14]

We agree with the conclusions of Jarvis, *et al,* that cotinine measurements from blood, saliva, or urine are applicable to a range of issues requiring validation of nicotine intake or cessation of tobacco use. Current data from our laboratory** do not support our previous finding of sustained saliva cotinine levels following cessation of smoking and demonstrate similar elimination times for cotinine from various body fluids. The elimination times of cotinine in passive smokers, however, remain longer than those observed in cigarette smokers.

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*Haley NJ, Sepkovic DW, Louis E, Hoffmann D: Absorption and elimination of nicotine by smokers, nonsmokers and chewers of nicotine gum. Presented at the International Symposium on Nicotine, Brisbane, Australia, 1987.

**Haley NJ, Sepkovic DW, Hoffmann D: Elimination of cotinine from body fluids: Nicotine disposition in smokers, nonsmokers, and chewers of nicotine gum. Paper submitted to Am J Public Health.

Response from Jarvis, *et al.*

We are grateful for the opportunity to respond to the letter from Drs. Sepkovic and Haley commenting on our recent study.[1] Our main purpose was to compare rates of decline of concentrations of cotinine in different body fluids. We undertook the study because Sepkovic and Haley reported that saliva samples were less suited than plasma or urine for validating claims of smoking cessation.[2] We are glad that they now agree that the rate of

decline of cotinine concentration in saliva is similar to that in plasma and urine, and that their earlier results were incorrect.

Drs. Sepkovic and Haley question our use of nicotine capsules as a source of cotinine in nonsmokers because it fails to consider the lung as a site of metabolism. However, this is not an issue that affects our study, since we were not studying nicotine metabolism. Nicotine capsules were chosen simply as a means of achieving high cotinine levels. At least 12 hours elapsed after the last nicotine dose before cotinine sampling began. Twelve hours after smoking cessation, very little nicotine is expected to remain in the lung, so our subjects are similar to abstinent smokers in this respect. In addition, Benowitz, *et al,*[3] found the half-life of cotinine to be similar for subjects comparing smoking cessation to after intravenous cotinine conditions, indicating that nicotine metabolism has little impact.

Possible half-life differences between smokers and nonsmokers are important for interpretation of passive smoking dosimetry. To address this issue, one must consider two comparisons: 1) Is there a difference in the rate of metabolism of cotinine in smokers versus nonsmokers?; 2) Is there a different impact of continuing generation of cotinine in abstinent smokers versus passive smokers?

Our data indicate that, at comparable concentrations, the half-lives of cotinine are similar in nonsmokers to those reported in smokers in other studies. Kyerematen, *et al,*[4] did report differences between smokers and nonsmokers, but the magnitude of the difference was small (13 versus 10 hours). Thus, we conclude that at comparable blood concentrations the half-lives of cotinine, and presumably the rate of metabolism, are similar or only slightly different comparing smokers and nonsmokers.

The second question—the impact of continuing generation of cotinine in smokers versus passive smokers—remains to be answered. Etzel, *et al,*[5] and Haley, *et al,** report that in infants and adults, respectively, the half-life of cotinine is longer in passive smokers than in smokers. In contrast, it was noted by Haley, *et al,** that half-lives of cotinine were similar in abstinent smokers and ex-smokers after the latter had chewed nicotine gum.

These observations suggest that the longer half-life of cotinine in people passively exposed to tobacco smoke has nothing to do with different rates of metabolism but rather is due to continued introduction of cotinine into the circulation from ongoing low level exposure or from slow release of nicotine from tissue stores. In either case, continuing generation of cotinine from nicotine would prolong the half-life of cotinine in passive smokers, but would have no impact on half-life at the high levels of cotinine seen in smokers or in nicotine gum chewers.

*Haley NJ, Sepkovic DW, Louis E, Hoffmann D: Absorption and elimination of nicotine by smokers, nonsmokers and chewers of nicotine gum. Presented at the International Symposium on Nicotine, Gold Coast, Australia, 1987.

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EFNEP (Expanded Food and Nutrition Program)

I am writing in regard to the "Letters to the Editor" column in the January 1988 issue,[1] in which Barbara C. Sterne addresses an article on the relationship of participation in food assistance programs to the nutritional quality of diets published in the July 1987 issue.[2] She brings up an important point—that education on how to get the most nutrition for the food dollar "is sadly lacking in all but the WIC program." However, she has overlooked EFNEP (Expanded Food and Nutrition Program)—a nutrition education program administered out of the Extension Service at the county level. EFNEP, through trained paraprofessionals, works with low-income persons with young children (young families) on an intensive basis toward the goals of causing positive behavior change and acquisition of new food-related skills. EFNEP works with WIC and is able to provide the long-term intensive education that WIC cannot due to funding and staff restraints.

In Minnesota during 1987, EFNEP:

- reached 2,307 low/limited income participants and 8,070 family members;
- reached 3,414 low/limited income youth in 241 youth groups;
- utilized 441 volunteers;
- ensured that 60% of EFNEP participants received Food Stamps and 51% received WIC;
- demonstrated an average 43% knowledge increase occurred as a result of information taught; and
- found that more than 90% of participants exhibited a more varied diet as a result of EFNEP participation.

Statistics alone do not tell the whole EFNEP story. The self-sufficiency and self-esteem that some low-income persons receive from EFNEP is invaluable. Readers who would like additional information on Minnesota's EFNEP may contact me at 612/624-7479.

Editor's Note: For *national EFNEP information,* contact Extension Service, US Department of Agriculture, Office of Home Economics and Human Nutrition 202/447-2908. At the *local level,* EFNEP is administered through the Director of the Cooperative Extension Service, located at the land-grant university in the various states.

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Response from B. C. Sterne

In response to Ellen Schuster's letter regarding the Expanded Food and Nutrition Education Program (EFNEP), I certainly did not mean to imply that there are no other nutrition education programs available for fami-